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APPLICATION NO.	FILING DATE	FIRST NAMED INVENTOR	ATTORNEY DOCKET NO.	CONFIRMATION NO.
10/695,089	10/28/2003	Wing Y. Cheung	15039-2	4436

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EXAMINER	
KAPUSHOC, STEPHEN THOMAS	
ART UNIT	**PAPER NUMBER**
1634	

DATE MAILED: 08/28/2006

Please find below and/or attached an Office communication concerning this application or proceeding.

Office Action Summary	Application No.	Applicant(s)	
	10/695,089	CHEUNG ET AL.	
	Examiner	Art Unit	
	Stephen Kapushoc	1634	

-- The MAILING DATE of this communication appears on the cover sheet with the correspondence address --

Period for Reply

A SHORTENED STATUTORY PERIOD FOR REPLY IS SET TO EXPIRE 3 MONTH(S) OR THIRTY (30) DAYS, WHICHEVER IS LONGER, FROM THE MAILING DATE OF THIS COMMUNICATION.

- Extensions of time may be available under the provisions of 37 CFR 1.136(a). In no event, however, may a reply be timely filed after SIX (6) MONTHS from the mailing date of this communication.
- If NO period for reply is specified above, the maximum statutory period will apply and will expire SIX (6) MONTHS from the mailing date of this communication.
- Failure to reply within the set or extended period for reply will, by statute, cause the application to become ABANDONED (35 U.S.C. § 133). Any reply received by the Office later than three months after the mailing date of this communication, even if timely filed, may reduce any earned patent term adjustment. See 37 CFR 1.704(b).

Status

1)☒ Responsive to communication(s) filed on *28 June 2006*.

2a)☐ This action is **FINAL**. 2b)☒ This action is non-final.

3)☐ Since this application is in condition for allowance except for formal matters, prosecution as to the merits is closed in accordance with the practice under *Ex parte Quayle*, 1935 C.D. 11, 453 O.G. 213.

Disposition of Claims

4)☒ Claim(s) *1-5* is/are pending in the application.

4a) Of the above claim(s) _____ is/are withdrawn from consideration.

5)☐ Claim(s) _____ is/are allowed.

6)☒ Claim(s) *1-5* is/are rejected.

7)☐ Claim(s) _____ is/are objected to.

8)☐ Claim(s) _____ are subject to restriction and/or election requirement.

Application Papers

9)☐ The specification is objected to by the Examiner.

10)☐ The drawing(s) filed on _____ is/are: a)☐ accepted or b)☐ objected to by the Examiner.

Applicant may not request that any objection to the drawing(s) be held in abeyance. See 37 CFR 1.85(a).

Replacement drawing sheet(s) including the correction is required if the drawing(s) is objected to. See 37 CFR 1.121(d).

11)☐ The oath or declaration is objected to by the Examiner. Note the attached Office Action or form PTO-152.

Priority under 35 U.S.C. § 119

12)☐ Acknowledgment is made of a claim for foreign priority under 35 U.S.C. § 119(a)-(d) or (f).

a)☐ All b)☐ Some * c)☐ None of:

1.☐ Certified copies of the priority documents have been received.

2.☐ Certified copies of the priority documents have been received in Application No. _____.

3.☐ Copies of the certified copies of the priority documents have been received in this National Stage application from the International Bureau (PCT Rule 17.2(a)).

* See the attached detailed Office action for a list of the certified copies not received.

Attachment(s)

1) ☒ Notice of References Cited (PTO-892)
2) ☐ Notice of Draftsperson's Patent Drawing Review (PTO-948)
3) ☐ Information Disclosure Statement(s) (PTO-1449 or PTO/SB/08) Paper No(s)/Mail Date _____.
4) ☐ Interview Summary (PTO-413) Paper No(s)/Mail Date. _____ .
5) ☐ Notice of Informal Patent Application (PTO-152)
6) ☐ Other: _____.

DETAILED ACTION

Claims 6-11 have been cancelled by the Amendment of 06/28/2006.
Claims 1-5 are pending and examined on the merits.

This Office Action is in reply to applicants' correspondence of 06/28/2006. Claims 6-11 have been cancelled.

Applicants' arguments and Declaration have been fully considered.

Any rejections or objections not reiterated herein have been withdrawn. This Office Action presents new grounds of rejection and is NON-FINAL.

Claim Rejections - 35 USC § 103

1. The following is a quotation of 35 U.S.C. 103(a) which forms the basis for all obviousness rejections set forth in this Office action:

> (a) A patent may not be obtained though the invention is not identically disclosed or described as set forth in section 102 of this title, if the differences between the subject matter sought to be patented and the prior art are such that the subject matter as a whole would have been obvious at the time the invention was made to a person having ordinary skill in the art to which said subject matter pertains. Patentability shall not be negatived by the manner in which the invention was made.

2. Claims 1-5 are rejected under 35 U.S.C. 103(a) as being unpatentable over Rutledge et al (1991, as cited in the IDS) in view of Hattori et al (1995, as cited in the IDS), and Sathasivan et al (1991).

Rutledge et al teaches the nucleic acid and deduced amino acid sequence of the *Brassica napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). The reference teaches that DNA was isolated from leaf nuclei, relevant to claim 1 step (a). The reference also teaches that imidazolinone herbicides act through inhibition of AHAS (p.39, left column, last paragraph), and further teaches that herbicide resistance in *B. napus* mutants results from two unlinked alleles, and that the effect of combining the alleles in a hybrid line is additive for imidazolinone resistance. The reference teaches that the

imidazolinone resistance alleles correspond to AHAS1 and AHAS3 (p.39, left column, last paragraph), and concludes that the sequences of the AHAS genes provides the basic information essential for the analysis of *Brassica* mutants with resistance to herbicides that act on AHAS (p.39, right column, last paragraph).

Rutledge et al does not indicate the nature of the mutations in AHAS 1 (PM1) and AHAS3 (PM2) that confer resistance to imidazolinone.

Hattori et al 1995 teaches the analysis of the AHAS3 gene from imidazolinone-resistant mutant *B. napus* cells, relevant to claim 1 step (c) of the instant application. The reference teaches that the AHAS3 gene from the mutant cells was cloned and sequenced, and the sequence of the gene from the mutant was compared to the wild-type AHAS3 sequence (p.420, right column, l.28). Hattori teaches the identification of a single basepair change (G to T) in AHAS3 that predicts a tryptophan to leucine amino acid change (p.421, left column, last paragraph), and provides a comparative alignment of deduced amino acid sequences in the region of the AHAS3 mutation responsible for herbicide resistance (p.421 Fig. 2). Based on the alignment provided in Fig. 2, and the sequence of the AHAS3 gene provided by Rutledge et al, it is evident that the G to T mutation taught by Hattori is equivalent to the PM2 mutation claimed in the instant application. Hattori concludes that the identified mutation site in the AHAS3 gene is involved in the binding of imidazolinone herbicides, and teaches that the recovery of the same mutation in tobacco and *B. napus*. Further relevant to claim 4 of the instant application, Hattori teaches the amplification of the AHAS1 gene from isolated genomic DNA prior to determining whether or not mutations are present.

Sathasivan et al teaches the analysis of an *A. thaliana* mutation in the acetolactate synthase gene (referred to within the reference as ALS, which is an art recognized synonym for AHAS). The reference teaches that the mutation provides the molecular basis for imidazolinone resistance in *A. thaliana* (p.1044 – Abstract). Sathasivan et al teaches the specific nature of the *A. thaliana* mutation responsible for herbicide resistance as a G to A single-point mutation at nucleotide 1958 of the coding sequence, which predicts a serine to asparagine substitution at amino acid 653 (p.1044, left column, last paragraph; Fig. 2; Table 1). Based on the nucleic acid sequence provided by Sathasivan et al (Fig 2B), the sequence of the AHAS1 gene provided by Rutledge et al as well as the teachings of Rutledge et al that the imidazolinone resistance alleles correspond to AHAS1 and AHAS3, and the fact that Hattori et al teaches a mutation in AHAS3, it is evident that *A. thaliana* G to A mutation taught by Sathasivan is equivalent to the PM1 mutation claimed in the instant application. The reference also teaches that similar mutations at corresponding nucleotide positions of other acetolactate synthase genes can confer imidazolinone resistance (p.1049, left column, last paragraph). Further relevant to claim 5, Sathasivan et al teaches the analysis of the sequence of the acetolactate synthase gene using a chain termination method (p.1045 – Nucleic acid techniques; Fig. 2), which is a primer extension based method for sequencing that can detect single nucleotide polymorphisms.

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have combined the information and methods provided in the cited references to have created the claimed invention of a method to assay for the

presence or absence of the PM1 and PM2 mutations to determine the imidazolinone tolerance of a plant. One would have been motivated to develop such an assay to efficiently determine the relative level of herbicide resistance in a plant using molecular techniques based on the teachings of Rutledge et al, which teaches that combining resistance alleles in a hybrid line has an additive effect on resistance to imidazolinone. One would have had a reasonable expectation of success because the cited references teach both the general aspects of the properties responsible for imidazolinone resistance, as well as the specific molecular characteristics that confer imidazolinone herbicide resistance. Rutledge et al teaches that the two alleles responsible for imidazolinone resistance in a *B. napus* mutant correspond to the AHAS1 and AHAS3 genes, and that the effect of combining the alleles in a hybrid line is additive for imidazolinone resistance (p.39, right column, last paragraph). Rutledge et al further teaches the nucleic acid sequences and deduced amino acid sequences of the *B. napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). Hattori et al teaches the identification of a G to T (tryptophan to leucine) mutation in the *B. napus* AHAS3 gene responsible for imidazolinone resistance that is equivalent to the PM2 mutation of the instant application. Sathasivan et al teaches the identification of a G to A (serine to asparagine) mutation in the *A. thaliana* ALS gene and provides a nucleic acid sequence indicating that this mutation is equivalent to the PM1 mutation of the instant application. It would be obvious to look for a G to A mutation at the PM1 position of the AHAS1 gene based on the teachings of Rutledge et al that there are two mutations that confer imidazolinone resistance and that one mutation is in AHAS1 and the other is in AHAS3

and the teachings of Hattori et al 1995 which teach that there is an imidazolinone resistance-conferring mutation in AHAS3 (which is identical to PM2). Thus based on Rutledge et al in view of Hattori et al 1995, it would be obvious to look for another mutation in the AHAS1 gene, and based on the teachings of Sathasivan et al it would be obvious to look for the G to A (Ser to Asn) mutation that is PM2.

Thus, in view of the teachings of the prior art, the claimed invention is prima facie obvious.

3. Claims 1-5 are rejected under 35 U.S.C. 103(a) as being unpatentable over Rutledge et al (1991, as cited in the IDS) in view of Hattori et al (1995, as cited in the IDS), and Hattori et al (1992).

Rutledge et al teaches the nucleic acid and deduced amino acid sequence of the *Brassica napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). The reference teaches that DNA was isolated from leaf nuclei, relevant to claim 1 step (a). The reference also teaches that imidazolinone herbicides act through inhibition of AHAS (p.39, left column, last paragraph), and further teaches that herbicide resistance in *B. napus* mutants results from two unlinked alleles, and that the effect of combining the alleles in a hybrid line is additive for imidazolinone resistance. The reference teaches that the imidazolinone resistance alleles correspond to AHAS1 and AHAS3 (p.39, left column, last paragraph), and concludes that the sequences of the AHAS genes provides the basic information essential for the analysis of *Brassica* mutants with resistance to herbicides that act on AHAS (p.39, right column, last paragraph). Further relevant to

claim 5, Rutledge et al teaches the analysis of the sequences of the AHAS genes using di-deoxy sequencing with primers (p.32 – DNA sequence analysis), which is a primer extension based method for sequencing that can detect single nucleotide polymorphisms.

Rutledge et al does not indicate the nature of the mutations in AHAS 1 (PM1) and AHAS3 (PM2) that confer resistance to imidazolinone.

Hattori et al 1995 teaches the analysis of the AHAS3 gene from imidazolinone-resistant mutant *B. napus* cells, relevant to claim 1 step (c) of the instant application. The reference teaches that the AHAS3 gene from the mutant cells was cloned and sequenced, and the sequence of the gene from the mutant was compared to the wild-type AHAS3 sequence (p.420, right column, l.28). Hattori teaches the identification of a single basepair change (G to T) in AHAS3 that predicts a tryptophan to leucine amino acid change (p.421, left column, last paragraph), and provides a comparative alignment of deduced amino acid sequences in the region of the AHAS3 mutation responsible for herbicide resistance (p.421 Fig. 2). Based on the alignment provided in Fig. 2, and the sequence of the AHAS3 gene provided by Rutledge et al, it is evident that the G to T mutation taught by Hattori is equivalent to the PM2 mutation claimed in the instant application. Hattori concludes that the identified mutation site in the AHAS3 gene is involved in the binding of imidazolinone herbicides, and teaches that the recovery of the same mutation in tobacco and *B. napus*. Further relevant to claim 4 of the instant application, Hattori teaches the amplification of the AHAS1 gene from isolated genomic DNA prior to determining whether or not mutations are present.

Hattori et al 1992 teaches the analysis of an imidazolinone resistance-conferring mutation in the AHAS gene of *A. thaliana*. The reference teaches that the mutation responsible for imidazolinone resistance is a G to A transition that predicts a Ser to Asn substitution in the amino acid sequence Ile Pro Ser Gly Gly (p.169 – Nucleotide sequence of imr1), and further teaches that the Ser that is substituted shows perfect conservation in all of the known wild-type plant AHAS genes including *B. napus*. provides the molecular basis for imidazolinone resistance in *A. thaliana* (p.1044 – Abstract). Based on the nucleic acid sequence (Fig 1C) and amino acid sequence context (Fig 1C; p.169 – Nucleotide sequence of imr1) provided by Hattori et al 1992, the sequence of the AHAS1 gene provided by Rutledge et al as well as the teachings of Rutledge et al that the imidazolinone resistance alleles correspond to AHAS1 and AHAS3, and the fact that Hattori et al 1995 teaches a mutation in AHAS3, it is evident that the *A. thaliana* G to A mutation taught by Hattori et al 1992 is equivalent to the PM1 mutation claimed in the instant application.

It would have been prima facie obvious to one of ordinary skill in the art at the time the invention was made to have combined the information and methods provided in the cited references to have created the claimed invention of a method to assay for the presence or absence of the PM1 and PM2 mutations to determine the imidazolinone tolerance of a plant. One would have been motivated to develop such an assay to efficiently determine the relative level of herbicide resistance in a plant using molecular techniques based on the teachings of Rutledge et al, which teaches that combining resistance alleles in a hybrid line has an additive effect on resistance to imidazolinone.

One would have had a reasonable expectation of success because the cited references teach both the general aspects of the properties responsible for imidazolinone resistance, as well as the specific molecular characteristics that confer imidazolinone herbicide resistance. Rutledge et al teaches that the two alleles responsible for imidazolinone resistance in a *B. napus* mutant correspond to the AHAS1 and AHAS3 genes, and that the effect of combining the alleles in a hybrid line is additive for imidazolinone resistance (p.39, right column, last paragraph). Rutledge et al further teaches the nucleic acid sequences and deduced amino acid sequences of the *B. napus* AHAS1 and AHAS3 genes (Fig. 2A and 2C). Hattori et al 1995 teaches the identification of a G to T (tryptophan to leucine) mutation in the *B. napus* AHAS3 gene responsible for imidazolinone resistance that is equivalent to the PM2 mutation of the instant application. Hattori et al 1992 teaches the identification of a G to A (Ser to Asn) mutation in the *A. thaliana* ALS gene and provides the amino acid sequence context of the alteration (i.e. Ile Pro Ser Gly Gly changed to Ile Pro Asn Gly Gly) indicating that this mutation is equivalent to the PM1 mutation of the instant application. It would be obvious to look for a G to A mutation at the PM1 position of the AHAS1 gene based on the teachings of Rutledge et al that there are two mutations that confer imidazolinone resistance and that one mutation is in AHAS1 and the other is in AHAS3 and the teachings of Hattori et al 1995 which teach that there is an imidazolinone resistance-conferring mutation in AHAS3 (which is identical to PM2). Thus based on Rutledge et al in view of Hattori et al 1995, it would be obvious to look for another mutation in the

AHAS1 gene, and based on the teachings of Hattori et al 1992 it would be obvious to look for the G to A (Ser to Asn) mutation that is PM2.

Thus, in view of the teachings of the prior art, the claimed invention is prima facie obvious.

Double Patenting

4. The nonstatutory double patenting rejection is based on a judicially created doctrine grounded in public policy (a policy reflected in the statute) so as to prevent the unjustified or improper timewise extension of the "right to exclude" granted by a patent and to prevent possible harassment by multiple assignees. A nonstatutory obviousness-type double patenting rejection is appropriate where the conflicting claims are not identical, but at least one examined application claim is not patentably distinct from the reference claim(s) because the examined application claim is either anticipated by, or would have been obvious over, the reference claim(s). See, e.g., In re Berg, 140 F.3d 1428, 46 USPQ2d 1226 (Fed. Cir. 1998); In re Goodman, 11 F.3d 1046, 29 USPQ2d 2010 (Fed. Cir. 1993); In re Longi, 759 F.2d 887, 225 USPQ 645 (Fed. Cir. 1985); In re Van Ornum, 686 F.2d 937, 214 USPQ 761 (CCPA 1982); In re Vogel, 422 F.2d 438, 164 USPQ 619 (CCPA 1970); and In re Thorington, 418 F.2d 528, 163 USPQ 644 (CCPA 1969).

A timely filed terminal disclaimer in compliance with 37 CFR 1.321(c) or 1.321(d) may be used to overcome an actual or provisional rejection based on a nonstatutory double patenting ground provided the conflicting application or patent either is shown to be commonly owned with this application, or claims an invention made as a result of activities undertaken within the scope of a joint research agreement.

Effective January 1, 1994, a registered attorney or agent of record may sign a terminal disclaimer. A terminal disclaimer signed by the assignee must fully comply with 37 CFR 3.73(b).

5. Claims 1-5 are provisionally rejected on the ground of nonstatutory double patenting over claims 1-25 of copending Application No. 10/695,546 (Pub. No.: US 2004/0171027 A1). This is a provisional double patenting rejection since the conflicting claims have not yet been patented.

The subject matter claimed in the instant application is fully disclosed in the referenced copending application and would be covered by any patent granted on that copending application since the referenced copending application and the instant application are claiming common subject matter, as follows:

6. The copending '546 application claims methods for assaying a *Brassica* plant for imidazolinone resistant comprising the steps of isolating genomic DNA from a plant and detecting the PM1 mutation in AHAS1 and the PM2 mutation in AHAS3. The claims of the copending application also encompass the amplification of the isolated DNA (relevant to claim 4 of the instant application), as well as methods to detect single nucleotide polymorphisms that utilize the extension of primers (relevant to claim 5 of the instant application). Although the copending '546 application cites different nucleotide positions (paragraphs [0028]-[0029]) of the G to A mutation in AHAS1 and the G to T mutation in AHAS3, which are PM1 and PM2 respectively, it is evident from a comparative alignment of the gene sequences in the copending applications that the identical mutations at the equivalent positions are claimed.

Furthermore, there is no apparent reason why applicant would be prevented from presenting claims corresponding to those of the instant application in the other copending application. See *In re Schneller*, 397 F.2d 350, 158 USPQ 210 (CCPA 1968). See also MPEP § 804.

Response to Remarks

Restriction Requirement and Claim Objections

Applicant has confirmed the election of Group I, claims 1-5 without traverse. Applicant submits that rejoinder of claims encompassing *B. capestris/rapa and B. juncea* is appropriate upon determination of allowable claims directed to *B. napus.* Further, applicant has traversed the Objection to the non-elected subject matter and supplied Somers et al (2002) and a reference to Bing et al (1996) indicating that use of the claimed sequences among the different *Brassica* species is obvious and known to those of ordinary skill in the art.

The Objection has been withdrawn, and rejoinder of claims encompassing *B. capestris/rapa and B. juncea* will be considered upon determination of allowable claims.

Requirement for information

In response to the Requirement for Information, Applicant has submitted a Declaration indicating that DNA-based assays for the presence of the PM1 And PM2 mutations were not publicly disclosed or sold prior to October 29, 2002. This declaration has been fully considered and satisfies the Requirement for Information.

Rejections pursuant to 35 USC 103

Applicant has traversed the rejection of claims 1-5 as unpatentable over Beetham et al in view of Rutledge et al, Hattori et al, and Sathasivan et al.

Applicant has traversed the rejection based on the teaching of Beetham et al of an S635N mutation in the AHAS3 gene which Beetham teaches as PM2 (p.3 and p.32 of Beetham). Applicant argues that this teaching contradicts the teachings of Hattori et al which teaches the different mutation (G to T / Trp to Leu in AHAS3), which is in fact the PM2 mutation of the instant application. This argument is found persuasive, because while Beetham et al does clearly teach the PM2 mutation of the instant application (p.7 lines 22-25), as well as mutations in both AHAS1 and AHAS 3 (p.3

Ins24-27), Beetham is not clear on the specific location of a Ser to Asn alteration in AHAS1.

The rejection of claims as unpatentable over Beetham et al in view of Rutledge et al, Hattori et al, and Sathasivan et al is withdrawn. A new rejection of claims 1-5 as unpatentable over Rutledge et al in view of Hattori et al and Sathasivan et al is presented.

Applicant argues that Hattori et al (1995) is deficient in any teachings of the PM1 mutations of *B. napus*. However, the Examiner has cited Hattori et al (1995) for its clear teaching of the molecular nature of the PM2 mutation in AHAS3, as recognized by Applicant (page 3 of remarks).

Applicant argues that Sathsivan et al is devoid of any teaching of the PM1 mutation of *B. napus* and is thus irrelevant to the claimed assay for the PM1 and PM2 mutations in *Brassica*. This is not found persuasive because, while Sathsivan et al uses a different plant (*A. thaliana* instead of *Brassica*), the reference clearly indicates a G to A alteration in the nucleic acid sequence of an AHAS gene resulting in a Ser to Asn substitution. And while Sathsivan et al does not specifically teach an analysis of *Brassica* per se, the reference does teach that site of the *A. thaliana* mutation is conserved in other plant species and thus may be implicated in herbicide resistance in other plants. Thus in view of the teachings of the other references (i.e. Rutledge et al which teaches that imidazolinone resistance mutations are in AHAS1 and AHAS3 and the sequences of the genes, and Hattori et al 1995 which teaches a specific imidazolinone resistance mutation in AHAS3 (that is PM2 of the instant application)), the teachings of Sathsivan et al are extremely relevant to the G to A (Ser to Asn) mutation in AHAS1 that is the PM1 mutation.

Applicant argues that the Examiner has used a hindsight reasoning to combine the references to arrive at the claimed invention because none of the cited references provide any motivation leading to the proposed combination. This is not found persuasive because Rutledge et al clearly indicates the involvement of two mutations, one in the AHAS1 gene and one in the AHAS3 gene, in imidazolinone resistance, as well as the additive effects of the two mutations together in a hybrid line (p.39, left col.,

last paragraph), and Rutledge et al also states that the provided information is essential for the analysis of *Brassica* mutants with herbicide resistance (p.39, right col., last paragraph). Thus there is clear motivation for the analysis of the particular mutations that are causative of imidazolinone resistance in *Brassica*.

No response to Double Patenting Rejection

It is noted that applicant has provided no Response concerning the Double patenting Rejection; the rejection is maintained. Applicant is advised that Response to all matters raised in the Office Action is required, otherwise a Response may be found Non-Responsive

Conclusion

No claim is free of the art, no claim is allowed.

Any inquiry concerning this communication or earlier communications from the examiner should be directed to Stephen Kapushoc whose telephone number is 571-272-3312. The examiner can normally be reached on Monday through Friday, from 8am until 5pm.

If attempts to reach the examiner by telephone are unsuccessful, the examiner's supervisor, Gary Jones can be reached at 571-272-0745. The fax phone number for the organization where this application or proceeding is assigned is 571-273-8300.

Information regarding the status of an application may be obtained from the Patent Application Information Retrieval (PAIR) system. Status information for published applications may be obtained from either Private PAIR or Public PAIR. Status information for unpublished applications is available through Private PAIR only. For more information about the PAIR system, see http://pair-direct.uspto.gov. Should you have questions on access to the Private PAIR system, contact the Electronic Business Center (EBC) at 866-217-9197 (toll-free).

Stephen Kapushoc
Art Unit 1634

Jehanne Sitton

JEHANNE SITTON
PRIMARY EXAMINER
8/21/06